

3. (Amended) The expression vector of claim 2, wherein said coding region encoding said secreted chemokine is expressed from an internal ribosome entry site.

8. (Twice Amended) The expression vector of claim 1, wherein said chemokine encoding region encodes a chemokine that binds to a C-C chemokine 5 receptor, a C-C chemokine 3 receptor, a C-C chemokine 1 receptor or a CXR4 receptor.

9. (Twice Amended) The expression vector of claim 1, wherein said chemokine encoding region encodes a chemokine that binds to a C-C chemokine 5 receptor.

10. (Twice Amended) The expression vector of claim 1, wherein said chemokine encoding region encodes a chemokine that binds to a C-C chemokine 3 receptor.

11. (Twice Amended) The expression vector of claim 1, wherein said chemokine encoding region encodes a chemokine that binds to a C-C chemokine 1 receptor.

12. (Twice Amended) The expression vector of claim 1, wherein said chemokine encoding region encodes a chemokine that binds to a CXR4 receptor.

17. (Twice Amended) An *ex vivo* method of inhibiting phenotypic expression of a chemokine receptor in a cell, wherein the method comprises blocking cell surface expression of said chemokine receptor by binding of said chemokine receptor with an intrakine.

18. (Twice Amended) The method of claim 17, further defined as comprising the steps of:

obtaining a vector comprising a nucleic acid segment encoding a promoter; an intracellular retention signal sequence and a chemokine receptor binding polypeptide coding region; and

transducing said vector into said cell;

wherein said vector expresses said intracellular retention signal sequence and chemokine receptor binding polypeptide coding region under the transcriptional control of said promoter to produce a fusion polypeptide when transduced into said cell.

23. (Twice Amended) An *ex vivo* method of inhibiting HIV infection of a cell, said method comprising phenotypically knocking out an HIV co-receptor in said cell by binding of said HIV co-receptor with an intrakine, wherein said phenotypic knock-out of said HIV co-receptor in said cell inhibits infection of said cell.

34. (Amended) The method of claim 24, wherein said cell is transduced with a CXC-chemokine coding region fused to an endoplasmic reticulum (ER)-retention signal to intracellularly block the transport and surface expression of an endogenous CXCR4 receptor.

38. (Twice Amended) A composition comprising the expression vector of claim 1 and a pharmaceutically acceptable solution.

39. (Twice Amended) A method of increasing white blood cell count in a subject with an HIV infection comprising administering to said subject a pharmaceutical composition comprising a lymphocyte, a monocyte, a macrophage or a stem cell transduced *ex vivo* with the vector of claim 1, thereby increasing white blood cell count in said subject with an HIV infection.

REMARKS

The present invention relates to novel methods and compositions for the treatment of HIV infection and for methods of conferring HIV resistance. The invention discloses, *inter alia*, methods of inhibiting HIV co-receptor cell surface expression using intracellular retained cytokines, *i.e.*, “intrakines,” to bind to the receptors intracellularly and prevent transport of the receptors to the cell surface. The invention further relates to inhibiting HIV-1 infection using intrakines and secreted chemokines to competitively inhibit HIV-1 from binding with a co-receptor.